REMARKS

Claims 1 and 3 are pending. The claims have been amended to more particularly specify a preferred embodiment. No new matter has been added. Reconsideration is requested.

Claim 3 was objected to, as appearing to have a typographical error in line 13. The claim has been amended to correct the error. Withdrawal of the rejection is respectfully requested.

Claims 1 and 3 were rejected under 35 USC § 112, first paragraph, as not being enabled. The Examiner has taken the position that the claims are overly broad, and that it would require undue experimentation to practice the invention as claimed. Although Applicants do not agree, in order to expedite prosecution, claims 1 and 3 have been amended to include three symptoms exhibited by the model mouse. Accordingly, the claims recite a model mouse, and a method for screening a remedy comprising administering a substance to a model mouse, subject matter that the Examiner indicated was enabled. It is respectfully submitted that claims 1 and 3 are free of the rejection. Reconsideration and withdrawal of the rejection are accordingly requested.

Claim 1 has been rejected under 35 USC § 103 as being obvious over Takai in view of Abbate or Kalluri. This rejection is traversed for the following reasons.

Comparison of the present invention and the citations

Takai (1996, Nature, Vol. 379, pages 346-348) discloses a method of producing a FcγRIIB receptor deficient mouse and the regulation system of Type I allergy. However, there is no suggestion regarding immunization with Type IV collagen and no disclosure regarding Goodpasture's syndorome nor Goodpasture's syndrome animal model.

Kalluri (1994, PNAS, Vol. 91, pages 6201-6205) discloses that Goodpasture's symdrome animal model obtained by being immunized with recombinant proteins shows weak alveolar hemorrhage, and hence are far from satisfactory to be used as an

appropriate animal model pulmonary lesion of Goodpasture's syndrome which is often lethal to humans.

Abbate (1998, Kidney International, Vol. 54, pages 1550-1561) discloses the animal model using a rat, not a mouse as in described in claims 1 and 3. As Abbate's animal model shows extremely weak alveolar hemorrhage, it is difficult to distinguish from other nonspecific inflammation images and the model is not appropriate for use. Also, the weakness of symptoms of alveolar hemorrhage and inflammation images leads difficulty in distinguishing from nonspecific bleeding or inflammation which is induced from immunological adjuvant itself in rare cases. Therefore Abbate's animal model is not appropriate for the Goodpasture's syndrome animal model.

Abbate and Kalluri describes that one of the antigens of Goodpasture's syndrome is in Type IV collagen. However, Goodpasture's symdrome animal models obtained in these methods show weak alveolar hemorrhage, and hence are far from satisfactory to be used as an appropriate animal model pulmonary lesion of Goodpasture's syndrome which is often lethal to humans.

Claim 1 has been amended to recite:

"A model mouse showing symptoms of diffuse alveolar hemorrhage, glomerulonephritis, and the appearance of antikidney glomerular basement membrane antibody," and the model mouse having three the phenotypes is defined as a claimed object.

The model mouse, which has three phenotypes, showing the severe symptom of the Goodpasture's symdrome is unpredictable, even if the three of Takai, Abbate and Kalluri are taken into consideration together. In conclusion, it is respectfully submitted that claim 1 is not obvious in view of the cited references. Reconsideration and withdrawal of the rejection under 35 USC § 103 are respectfully requested.

All rejections having been addressed, it is respectfully submitted that this application is in condition for allowance, and Notice to that effect is respectfully requested.

Respectfully submitted,

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